Short report

Bilateral cerebellar dysfunctions in a unilateral meso-diencephalic lesion

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SUMMARY The clinical syndrome of a 65-year-old patient with a slit-shaped right-sided meso-diencephalic lesion was analysed. A cerebellar syndrome with limb-kinetic ataxia, intention tremor and hypotonicity in all extremities as well as ataxic dysarthria was found. The disruption of the two cerebello-(rubro)—thalamic pathways probably explained the signs of bilateral cerebellar dysfunction. The uncrossed ascending limb of the right, and the crossed one of the left brachium conjunctivum may have been damaged by the unilateral lesion extending between caudal midbrain and dorsal thalamus.

Most of the fibres which constitute the superior cerebellar peduncle leave the cerebellum and originate in cells of the dentate nucleus but also arise from neurons of the globose and emboliforme nuclei. The crossed ascending fibres of the brachia conjunctiva constitute the major outflow from the cerebellum, they form the cerebello-(rubro)-thalamic and dentato-thalamic tracts. Lesions of the superior cerebellar peduncle lead to the same abnormalities as are seen following destruction of hemispheric portions of the posterior cerebellar lobe. Monkeys with section of both cerebellar peduncles show a considerable ataxia, hypotonicity and marked action tremor. We report a patient with bilateral cerebellar dysfunction, caused by a unilateral meso-diencephallic lesion possibly affecting both superior cerebellar peduncles.

Case report

On 31 January 1979 a 65-year-old white male suddenly became unconscious. After several minutes he regained consciousness. He seemed confused and was not able to utter a sound, although he understood simple commands such as “open your eyes,” or “do you want something to drink?” He was admitted to a general hospital where neurological examination showed bilateral miosis, convergent strabism, vertical gaze paresis on upward gaze with gaze-paretic nystagmus, flaccid sensori-motor hemiparesis with increased stretch reflexes and Babinski sign on the left side, and dysmetric movements of the right upper extremity. The CT scan showed an acute haemorrhage in the right mesodiencephalic area. On 19 February 1979 the patient was admitted to our department. We found an isolated paresis of the left superior oblique muscle (the bilateral miosis, convergent strabism and paresis of upward gaze had disappeared). There was a sharp contrast between the monoplegia of the left arm, the slight motor deficit in the left leg, and the normal face. Stretch reflexes were increased, and Babinski's sign was present, on the left side; stretch reflexes on the right side were reduced. Appreciation of touch was reduced on the left upper extremity sparing the face; appreciation of pain was diminished in the left arm and face; positional sense was lost in the left hand only; vibratory sense and appreciation of temperature was impaired on the entire left half of the body. Tone was markedly reduced in all extremities; elbow- and knee-joints could easily be hyper-extruded. Because of “weakness” the patient could not keep himself upright without support. He was not able to walk because of a severe gait ataxia. Coarse dysmetric movements of the non-paretic extremities rendered the patient completely helpless.

By March 1979 the weakness of the left arm had improved, but dysmetria movement was evident, and there was bilateral coarse intention tremor and pathological rebound. Verbal communication was hampered by dysarthric and dysphonic speech; the
voice was breathy with voice breaks and diplophonic intervals. Articulation was characterised by a marked slowness of articulatory movements, reduced tightness of constrictions and the inability to achieve high tongue elevation in the production of vowels and consonants. Features of disordered prosody were slow speech rate, reduced modulation of pitch and intensity as well as an equalisation of syllable length. Furthermore, excess and equal stress on ordinarily unstressed syllables occurred. There was, however, no indication of an aphasic disorder. Apart from a grotesque ataxic macrographia, handwriting was not disturbed. Subsequently the patient complained of burning pain in the left arm. Clinical examination revealed hyperpathia of the left arm sparing the hand. This symptom disappeared within a few days.

Selective angiography of the left and right vertebral arteries revealed no abnormality. Repeated EEGs did not show any abnormality. The CSF was normal. CT-scan in March 1979, showed a small hypodense lesion in the right meso-diencephalic region (figure).

In the figure six details of the meso-diencephalic area are shown in a sequence from left-up to right-down. The upper left detail corresponds to a “section” of the brain stem through the inferior colliculi (at the transition to the rostral pons). At the level of the

Figure  CT scan. Six details of the meso-diencephalic area in a sequence from left up to right down. The arrows indicate the site of the lesion.
superior cerebellar left affected in the and dysmetria had been left superior to the decussation.3

Left superior of the cerebellar by ported in side dysmetria its the superior lessened the homolateral to the lesion reached the rostral midbrain, explaining this patient; the crossed ascending limb of the left superior cerebellar peduncle should account for the occurrence of ataxic dysarthria. According to these authors the dominance of the left cerebellar hemisphere in the regulation of speech may derive from access of this hemisphere to the nondominant (right) cerebral hemisphere. The access of cerebellar efferent fibres to the right cerebral hemisphere has certainly been disrupted in our patient.

References

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